result of identifying the target molecules of ginsenoside Rb_1 or its metabolites, novel compounds which can modify the functions of the target molecules, would be synthesized. Then the development of remedies for spinal cord injuries, neuronal traumatic injuries or traumatic injuries can be directed.

Further, glial cells especially oligodendrocytes are known to enter apoptosis in cases of spinal cord injuries. As a result, demyelination occurs to deteriorate or aggravate the neural symptoms associated with spinal cord injuries (Crowe, M. J. et al., Nature Med. 3, 73-76, 1997; Emery, E. et al., J. Neurosurg. 89, 911-920, 1998). The experimental results in which intravenously administered ginsenoside Rb_1 significantly ameliorates paralysis or paraplegia of both hindlimbs of rats with spinal cord injuries, indicate that ginsenoside Rb_1 inhibits apoptosis or apoptosis-like cell death of oligodendrocytes and thereby ameliorates the symptoms of spinal cord injuries. Consequently, ginsenoside Rb_1 of the present invention is thought to be useful for prevention, therapy or treatment of brain and nervous diseases accompanied by demyelination (multiple sclerosis, Binswanger's dementia, etc.). Further, the experimental results, in which intravenously administered ginsenoside Rb_1 ameliorates paralysis of both hindlimbs of rats with spinal cord injuries (paraplegia), suggest that injured nerve fibers or nervous tissues can be regenerated as a result of administering

ginsenoside Rb_1.

The experimental results described above have demonstrated that the preparations for intravenous administration comprising ginsenoside Rb_1 or its salt can induce regeneration and/or reconstruction of the damaged or reduced cerebrovascular networks in cases of cerebral apoplexy. The preparations have also been shown to exhibit a protective action on brain cells (including glial cells) and a protective action on nerve cells (JP98/365560 and PCT/JP99/02550: "Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1"). As a result, the preparations protect brain tissues. It has also been demonstrated that the preparations for intravenous administration comprising ginsenoside Rb_1 or its salt inhibit not only the primary lesions in the nervous tissues, but also the secondary lesions in brain regions which have synaptic connections (fiber connections) with the primary lesions. Further, the pharmaceutical composition comprising ginsenoside Rb_1 is expected to be an epoch-making remedy for spinal cord injuries and neural traumatic injuries (neurotrauma), as well as exhibiting efficacy and effectiveness against traumatic injuries to the peripheral tissues.

Ginsenoside Rb_1, or its salt, is known to be a component of medicinal ginseng and is very low in toxicity.

Examples

The present invention will be explained in detail by concrete examples, but the present invention is not limited within these examples.

Example 1 (Experiment on intravenous infusion of ginsenoside Rb_1)

Male SH-SP rats, at the age of 12-13 weeks, (stroke-prone spontaneously hypertensive rat: weighing 250 - 300 g), were used. Animals were housed in an air-conditioned room furnished with a 12:12 hour light-dark cycle, and water and feeds were supplied ad libitum. The mean blood pressure of the animals was 203±6.9 mmHg. The following experiments were conducted in accordance with the Guide for Animal Experimentation at Ehime University School of Medicine. The cortical branch of the left middle cerebral artery (MCA) of SH-SP rats were coagulated and cut, while their rectal temperature was maintained at 37±0.2°C under inhalation anesthesia.

Immediately after MCA permanent occlusion, 60 μl of physiological saline containing ginsenoside Rb_1 at a concentration of 1 μg/μl or 0.1 μg/μl (60 μg or 6 μg as ginsenoside Rb_1) was injected once into the left femoral vein. Then a catheter connected to the Alza osmotic minipump implanted subcutaneously in the back of each animal was inserted into the same vein from the point of the single injection of ginsenoside